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This is a chapter describing medical therapy methods for food poisonings. These include Staphylococcal enterotoxemia and other bacterial enterotoxemias due to contamination of food by preformed toxins, botulism, mushroom poisoning and other poisons of plant origin, poisons of fungus origin including ergotism and mycotoxin poisoning, shellfish poisoning and poisons from other aquatic animals, gastroenteritis due to toxin secreting or invasive bacteria.		

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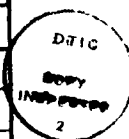
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FOOD POISONING

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FOOD POISONING

method of
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The foodborne illnesses comprise a wide variety of toxemias and infections. Their common denominator is the transmission of a causative agent via food. This assortment of maladies can best be subdivided into three major categories.

The first category includes the traditional forms of food poisoning caused by preformed toxins that may contaminate different dietary constituents. These toxins may be of biologic origin (microbial, plant, or animal) or they may be inorganic or synthesized organic substances.

The second category includes the many forms of acute gastroenteritis caused by pathogenic microorganisms. For inclusion in this category, local gastrointestinal manifestations must occur. Symptoms caused by some organisms are due to invasion of the gut mucosa, while other organisms cause illness by releasing toxins into the gut lumen.

The last category includes the microorganisms that cause generalized nonintestinal infectious illnesses despite the fact that the gut serves as the portal of entry into the body. This group includes viruses such as those causing poliomyelitis or hepatitis, bacteria causing tuberculosis or brucellosis, rickettsia causing Q fever, or parasites causing trichinellosis or toxoplasmosis. Therapy for these diverse generalized infections is covered in other chapters of the Infectious Diseases section.

DIAGNOSTIC REQUIREMENTS

Several hundred outbreaks of foodborne illness are reported each year, and many others go unreported because they are relatively innocuous and of short duration. To approach the diagnosis of an illness:

1. Characterize the illness according to symptoms, physical findings, and degree of severity.

2. Obtain an epidemiologic history to determine if similar illness has occurred concomitantly in companions or family members. Seek to identify the contaminated food in terms of when, what, and where it was eaten. In large outbreaks after a common meal, attack rates can be calculated for each food to identify the offending item. Contact the local Health Department to help conduct the investigation and process diagnostic specimens.

3. Obtain specimens for laboratory analysis. Recent food items, vomitus, stool, urine, serum, and unclotted blood samples may be of value for toxicologic screening or specific cultures. Examination of diarrheic stool may reveal leukocytes that suggest an invasive enteritis, or the presence of parasites or their cysts. A rapid diagnosis of viral diarrhea can be made by electron microscopy using direct negative contrast stains, if the facilities are available.

4. Routine blood counts and urinalysis are rarely helpful to establish a diagnosis. Serum electrolyte measurements are important to characterize body fluid derangements, and blood gas values are valuable in patients with progressive neurologic signs or overt respiratory difficulty.

FOOD POISONING

Food may contain or be contaminated by a wide variety of toxins. Man-

agement of poisoning due to these toxins is ultimately dependent upon recognition of cause and the combined use of specific antidotes and general supportive measures. Certain food poisonings produce a true medical emergency and treatment must be initiated without delay. Acute gastrointestinal symptoms herald the onset of most, but not all, kinds of food poisoning. In other forms, neurotoxicity may be the principal manifestation, and respiratory failure the cause of death. Shock, hemolysis, or failure of vital organs can also occur.

General Concepts

PRINCIPLES OF MANAGEMENT. The overall management of food poisoning includes the following points:

1. Monitor vital signs initially and on a continuing basis until the danger of shock or respiratory paralysis is over. Give these complications first priority in treatment.
2. Determine the source and type of food poisoning for planning specific therapy, for estimating prognosis and the need for hospitalization, and for anticipating complications.
3. Use specific therapy if indicated.
4. Replace fluid and electrolyte losses and maintain homeostatic balances thereafter. Oral sugar-electrolyte solutions may be effective, especially in children with mild diarrhea (see article on Parenteral Fluid Therapy for Infants and Children, Section 76?).
5. Correct and control hypoglycemia, if present.
6. Eliminate unabsorbed toxins from the gut if severe vomiting has not occurred. Gastric lavage, enemas, and a sodium sulfate purgative, 15 grams in 300 ml water instilled via the lavage tubing, can be of value if

used promptly in adults. Ipecac syrup, 15 to 20 ml orally, or apomorphine hydrochloride, 6 mg subcutaneously, may serve as an emetic, but should not be used if tracheal aspiration seems a likely possibility.

7. Hospitalize poisoned infants and children, since their therapy is often difficult to manage.

8. Provide general supportive care and symptomatic therapy. Nausea and vomiting may be alleviated in adults by prochlorperazine (Compazine), 5 or 25 mg by rectal suppository or 10 mg intramuscularly. As an alternative in adults, trimethobenzamine hydrochloride (Tigan), 200 mg, may be used by either route. Drugs for treating diarrhea in adults include paregoric, 4 ml every 2 hours; kaolin with pectin (Kaopectin), 30 ml every hour; diphenoxylate hydrochloride with atropine sulfate (Lomotil), 5 mg every 6 hours; or loperamide hydrochloride (Imodium), 8 mg followed by 2 mg after each liquid stool for a maximum daily total of 16 mg. However, drugs which slow the intestinal transit time should not be used for diarrhea caused by organisms that penetrate the gut mucosa. Propantheline (Pro-Banthine), 15 mg, may be given orally or intramuscularly as an antispasmodic. Codeine, 60 mg, or meperidine (Demerol), 100 mg, is useful for pain and can be given intramuscularly. Most pediatricians, however, advise against using such drugs for young children with acute vomiting or diarrheic illnesses.

DIFFERENTIAL DIAGNOSIS. Food poisoning must be differentiated from an acute surgical abdomen, acute infections of the intestinal tract, and the accidental contamination of food with heavy metals, chemicals, pesticides, or other poisonous industrial products. Toxin centers are currently available in many areas to provide rapid information. These should be contacted by phone. Also, see the article on Acute Miscellaneous Poisoning (Section ?16?).

Preformed Toxins of Bacterial Origin

Enterotoxemia

Protein toxins produced during the growth of Staphylococcal aureus in food cause the most common form of food poisoning. This may occur simultaneously in many people at camps, picnics, and the like, and thereby produce a mini-"mass casualty" situation. Symptoms begin 4 to 8 hours after ingestion of toxin and include severe retching, vomiting, diarrhea, and cramping abdominal pain. Intoxication is self-limited and generally benign. Shock can occur, however, most often in patients at the extreme ages of life. A similar preformed enterotoxin is produced by Bacillus cereus; it is most often found in reheated rice. Clostridium perfringens food poisoning is also relatively common. The A- and C- type strains produce enterotoxin when the bacteria sporulate. Clostridial toxins develop most often in meat dishes served a second time. In contrast, toxigenic Eschericia coli also produce enterotoxin, but only after they have first colonized the bowel.

Therapy is nonspecific in patients poisoned by preformed enterotoxins in food, and antibiotics are contraindicated. Only patients with severe symptoms and dehydration need be hospitalized. Most patients do not lose enough fluid to require intravenous infusions. Replacement of losses can generally be accomplished by the oral route.

Botulism

Therapy of botulism is directed at the prevention or control of respiratory muscle paralysis. Sedatives should be avoided since they may mask developing neurologic symptoms. These often begin with diplopia, dry-

ness of the mouth, or dysphagia. If present, respiratory failure takes first priority in management.

Because neuromuscular paralysis may progress with unpredictable speed, the patient should be hospitalized in anticipation of possible need for tracheostomy and ventilatory support. Such therapy must be aggressive and include meticulous, long-term respiratory management and intensive medical and nursing care (see article on Acute Respiratory Failure, Section 2).

Antiserum is of greatest value when given before paralysis begins. It should be used as soon as possible in patients showing progressive paralysis and in those who require ventilatory assistance. A trivalent A,B,E antiserum of equine origin is available from the National Centers for Disease Control, along with diagnostic consultation. One may call for this help at (404)-329-3753 by day or (404)-329-3644 at night. A heptavalent (A,B,C,D,E,F,G) despeciated horse immunoglobulin is under development, but use of this potentially safer product has not as yet been approved for man.

Drugs have not yet proven useful for treating botulism. Guanadine was introduced as an investigational drug, but it is relatively ineffective in preserving respiratory function and it may induce central hyperirritability. Another drug, 4-aminopyridine, has been tried with questionable results in Britain. A similar investigational drug, 3,4-diaminopyridine, is less toxic and shows dramatic but temporary effects on nerve transmission in animals. Drugs such as these may eventually prove useful in man, at least for helping to sustain respiration in a patient during transport to a treatment center.

Poisons of Plant Origin

Mushroom Poisoning

A complex variety of toxins are produced by different species of

poisonous mushrooms. Depending on the mushroom ingested, symptoms vary in time of onset as well as in the type of toxicity.

Symptoms may begin within 10 minutes after ingestion if the mushroom contains muscarine. Peripheral anticholinergic effects include sweating and salivation, lacrimation and visual disturbances, severe abdominal pain with retching, and vomiting, dizziness, and confusion. The muscarine alone can be lethal unless treated with its specific antidote, atropine, which should be given without delay in 1 to 2 mg doses by intravenous or subcutaneous injection. Subsequent doses are then given at 2 to 6 hour intervals. With control of symptoms, the quantity and frequency of atropine doses can be reduced.

Few patients exhibit muscarine effects as the sole manifestation of mushroom poisoning. Intestinal irritants and disulfiram-like compounds can also cause the rapid onset of nausea, vomiting, flushing, and tachycardia. Therapy is symptomatic. Substances such as indole derivatives and ibotenic acid may initiate central nervous system effects including mania, delirium, and even convulsions. These can best be treated with a rapidly acting intravenous barbiturate such as sodium thiopental (Pentothal), 0.1 to 0.2 gm.

In addition, one should also anticipate the onset of delayed additional symptoms that result from the slower action of cyclic peptide toxins such as phalloidine, amanitine, mycotrophine, and helvallic acid. The delayed form of toxicity may also appear de novo 6 to 12 after mushrooms are eaten. Severe, acute abdominal pain, nausea, vomiting, and diarrhea are followed by the slowly progressive development of hepatic and renal failure, as well as by the onset of extensive capillary, myocardial, and central nervous system damage. No effective antitoxins are available; management must include the correction of dehydration, intensive care, and sup-

portive therapy. Opiates can be used for control of pain. Attempts to remove toxins by gastric lavage or to absorb them with charcoal are relatively ineffective. Some of the low molecular weight toxins may be recovered by early hemodialysis. The use of a continuing intravenous infusion of 10 per cent dextrose may reduce hepatic toxicity, but the value of such infusions or the use of exchange transfusions has not been established.

Other aspects of fluid and electrolyte balance must be supervised with care. Urine output should be monitored hourly and consideration given to the use of mannitol diuresis if output falls (see article on Acute Renal Failure, Section 28?). If the patient survives for several days, the late complications are those of hepatorenal failure and infection by opportunistic microorganisms

Favism

Some persons with an inherited deficiency of red cell glucose-6-phosphate dehydrogenase experience a hemolytic crisis of varying severity after ingesting "broad beans" (Vicia fava). Mild shock can develop, but severe constitutional disturbances and renal failure are infrequent. Transfusions of packed red blood cells should be considered if hemolysis is massive. Corticosteroids are of undetermined value but may be given.

Lathyrism

Inclusion of Lathyrus peas in the diet may cause spasms, cramps, and weakness in the legs, and lead to degeneration of posterolateral tracts in the dorsolumbar spinal cord. This is accompanied by muscle paralysis and incontinence. Muscle relaxants are without value. Subcutaneous neostig-

mine methysulfate (Prostigmine), 0.5 mg given daily, has proven beneficial, especially if symptoms are of short duration.

Solanine Poisoning

This toxin is present in the eyes, sprouts, and skin of potatoes; it is generally destroyed by boiling. Symptoms include abdominal pain, vomiting and diarrhea, plus central nervous symptoms including confusion, hallucinations, and visual disturbances. Supportive care will be required for several days.

Oxalate Poisoning

Acute poisoning has been ascribed to the presence of oxalic acid in rhubarb leaves, beets, and spinach. Hypocalcemic tetany, if present, may be treated with intravenous calcium gluconate, 10 ml of a 10 per cent solution, repeated as required. The corrosive action of unabsorbed oxalic acid can be reduced by gastric lavage with an 0.15 per cent calcium hydroxide solution (lime water). Diuresis should be maintained by intravenous fluids to prevent deposition of calcium oxalate in renal tubules.

Poisons of Fungus Origin

Ergotism

Ergot alkaloids may be produced by Claviceps purpurea, a fungus that grows upon rye and other food grains. These alkaloids have multiple effects on adrenergic receptors of the smooth muscle of the uterus, blood vessels, and vasomotor centers. Treatment of severe arterial vasoconstriction may require paravertebral nerve block or a slow intravenous infusion of papaverine, 60 mg every 4 to 6 hours, to achieve vasodilation. Calcium

gluconate, 10 ml of a 10 per cent solution, may be used as an intravenous injection to relieve muscle pain.

Mycotoxin Poisoning

Moldy grains may contain fungi that secrete mycotoxins. Corn, peanuts, and grains in the warmer climates may be contaminated by one or more of the aflatoxins. These toxins can produce hepatic injury and intestinal bleeding in high doses, although such toxemia generally occurs in farm animals and fowl rather than in man. One potential long-term danger lies in the fact that aflatoxin B₁ is a potent hepatocarcinogen. Questions have been raised about a possible relationship of aflatoxin ingestion to the occurrence of liver carcinoma in Thailand.

Another major class of mycotoxins, the tricothecenes, are formed on grains in the colder climates. Flour produced from moldy grain has led to episodes of toxic aleukia, a protracted disease characterized by anemia, agranulocytosis, lymphopenia, vomiting, and bloody diarrhea. Tricothecene toxins are extremely stable and resistant to heating. They have complex actions on nucleic acid and protein synthesis comparable to those produced by ionizing radiation. The only known prophylactic measure for any of the agricultural mycotoxins is the avoidance of contaminated foodstuffs.

Poisons of Animal Origin

Shellfish Poisoning

Mussels and clams may accumulate heat-stable small molecular weight neurotoxins in their tissues by feeding upon plankton blooms during a so-

called "red tide". Saxitoxin stops neural transmission by blocking sodium channels in nerve fibers. Symptoms in man may appear within 10 minutes of eating poisonous shellfish. Initial paresthesia, giddiness, and ataxia may be followed swiftly by the onset of paralysis and difficulty in breathing. Full respiratory assistance must be provided aggressively without delay, for death can occur within minutes. The patient should be hospitalized for tracheostomy and artificial respiration. If life can be maintained for 24 hours, prognosis is good for survival without lasting effects. To diminish absorption of residual toxin, the stomach should be emptied by lavage and the gut purged. Supportive care includes maintenance of electrolyte, acid-base, and glucose homeostasis.

Fish Poisoning

Fish poisoning may be caused by a variety of toxins, many of which affect peripheral nerve transmission. Treatment must be based on symptomatic measures and the patients followed for the possible onset of shock or respiratory muscle failure.

Organs of the puffer fish contain tetrodotoxin, a neurotoxin having a mechanism of action similar to that of saxitoxin. If improperly cleaned fish are eaten, paralysis of nerve transmission and respiration will follow with the time of onset and severity being dependent upon dose. Treatment consists of immediate respiratory support as described for saxitoxin.

Ciguatoxins may appear in normally edible inshore fish throughout the Pacific and Caribbean. These toxins originate in algae and are transmitted through the food chain. Although ciguatoxins are not organophosphorus compounds, their anticholinesterase activity may be benefited by the use of a cholinesterase-reactivating drug such as pralidoxime chloride (Protopam). Such therapy may be considered for a trial if toxicity is life-threatening

and if blood cholinesterase values are low. The oxime is given intravenously in a total adult dose of 1 to 2 grams at the rate of 500 mg per minute (investigational). The dose may be repeated after 20 minutes.

On the other hand, neostigmine, which itself is a cholinesterase inhibitor, has been used with success in treating toxicity from poisonous barracuda.

Massive urticaria may accompany scombrotoxic fish (tuna, bonito, mackerel) poisoning. This should be treated with antihistamine drugs.

Other Food Poisons

Monosodium glutamate seasoning has been identified as the cause of "Chinese Restaurant Syndrome". Susceptible individuals may develop a brief, self-limited attack consisting of numbness, weakness, faintness and palpitation, sweating, lacrimation, and tightening of facial muscles. No specific therapy is indicated, but susceptibility should be recognized to prevent recurrence.

Toxic products from plants, such as white snakeroot, or solanines from the jimson weed, when ingested by cattle, may reach man via milk. Treatment consists of gastric lavage and intravenous glucose infusions.

The wide use of organophosphate insecticides has increased the likelihood that they may contaminate foodstuffs. Their acetylcholinesterase inhibition produces polyneuropathy, miosis, muscle fasciculations, manifestations of muscarine-like toxicity, and a reduction in blood cholinesterase activity. Pralidoxime chloride (Protopam), 1 gram intravenously, and atropine, 0.4 mg every 6 hours, may be combined for therapy. Persistent convulsions may call for the addition of trimethadione (Tridione), 1 gram (25

ml) orally every 15 minutes to a maximum of 5 grams, or sodium thiopental (Pentothal) intravenously, giving 0.1 to 0.2 grams in a 2.5 per cent solution.

ACUTE GASTROENTERITIS

Acute infections of the intestinal tract can be produced by bacteria, viruses, and protozoa. Bacteria must be able to traverse the normally acid milieu of the stomach, attach to intestinal mucosal cells by means of specialized pili or cell receptors, and colonize in the face of competition from indigenous gut organisms. These infections are most common during travel, especially to areas where unsanitary conditions permit fecal contamination of food and water.

a. Prophylactic measures include the practice of "avoidance" by selecting packaged or protected foods, using fluids that are unlikely to have been contaminated, and eating meals known to be prepared, held, and served under good hygienic conditions.

b. Drug prophylaxis may also be used under special, relatively brief circumstances. Bismuth subsalicylate (Pepto-Bismol) has been shown to have some prophylactic effectiveness against enterotoxigenic E. coli, Shigella, and rotavirus infections, although the recommended dose of 60 ml four times a day constitutes a sizable volume. Doxycycline (Vibramycin), 100 mg per day, may be protective against some enterotoxigenic E. coli, but many strains are resistant. Biweekly doses of 100 mg are only marginally effective. Since doxycycline is one of the tetracyclines, it may itself produce diarrhea or cause skin sensitization to sunshine in susceptible individuals. The drug combination of trimethoprim (80 mg) with sulfamethoxazole

(400 mg) in tablets (Septra), given twice daily, has prophylactic value against both enterotoxigenic E. coli and Shigella diarrheas if given for short periods of time. Septra, however, may induce a rash or anemia. Despite the partial effectiveness of prophylactic antibiotic regimens, many authorities argue against their use, citing the large incidence of diarrhea due to nonsusceptible organisms, the possibilities for side effects or adverse reaction, the masking of other bacterial infections, and the creation of antibiotic resistant strains.

c. Symptomatic therapy alone will suffice for the viral forms of enteritis and many of the bacterial diarrheas. Antiemetic drugs may provide welcome relief if nausea and vomiting are predominant. On the other hand, antiperistaltic medications are rarely, if ever, indicated. Antiperistaltic drugs are thought to retain toxins within the gut and to prevent their prompt evacuation. However, bismuth subsalicylate may help in the milder forms of enterotoxigenic diarrhea.

d. Drug therapy can include a five day course of Septra in the daily doses used for prophylaxis. Trimethoprim can be used alone in 200 mg doses twice a day for five days. Alternatively, a brief, three day course of a poorly absorbable antibiotic, bicozamicin, has recently proven effective therapy for acute travelers diarrhea of diverse causes when the antibiotic was given in oral doses of 500 mg four times daily.

For further information on the treatment of bacterial gastroenteritis, viral diarrheas, and parasitic diseases, the reader is referred to the separate articles on Cholera, Salmonellosis, and Typhoid Fever (Section 1) and Acute Infectious Diarrhea and Intestinal Parasites (Section 5).